Amendments to the Claims:

Please cancel claims 1-43 and add new claims 44-86. This listing of claims will replace all prior versions, and listings, of claims in the application:

Listing of Claims:

1-43. (cancelled)

44. (new) An agent for inhibiting at least one of release, maturation and replication of a member of the Flaviviridae family selected from Flavivirus, Pestivirus and Hepacivirus wherein the agent comprises, as an active component, at least one proteasome inhibitor in a pharmaceutical preparation.

45. (new) An agent as claimed in claim 44, wherein the agent inhibits at least one of release, maturation and replication of hepatitis C virus (HCV) and is used for the treatment and prophylaxis of HCV-induced hepatitides, flavivirus-induced fever, hemorrhages, leukopenia, thrombocytopenia, diarrheal diseases and encephalitides and also pestivirus-induced diseases.

46. (new) An agent as claimed in claim 45, wherein the proteasome inhibitor is a substance which inhibits, regulates or otherwise affects the activities of the ubiquitin/proteasome pathway; which specifically affects the enzymic activities of the complete 26S proteasome complex; and which specifically affects the enzymic activities of the free 20S, catalytically active, proteasome complex, which is not assembled with regulatory subunits.

47. (new) An agent as claimed in claim 45, wherein the proteasome inhibitor is taken up by higher eukaryotic cells and, after having been taken up into a cell, interacts with the catalytic subunits of the proteasome, and, in connection with this, blocks at least some of the proteolytic activities of the proteasome within the 26S or the 20S proteasome complex.

48. (new) An agent as claimed in claim 45, wherein in addition to proteasome inhibitors, the pharmaceutical preparation also comprises at least one further agent which affects, regulates or inhibits the cellular ubiquitin system, such as the activities of the ubiquitin-conjugating enzymes and/or of the ubiquitin-hydrolyzing enzymes.

49. (new) An agent as claimed in claim 45, wherein the proteasome inhibitor is administered in various forms *in vivo*, i.e. orally, intravenously, intramuscularly, subcutaneously or in encapsulated form, with or without cell specificity-carrying changes, which, due to using a particular administration and/or dose regime, exhibit low cytotoxicity, which do not elicit any side effects, or only elicit insignificant side effects, and which exhibit a relatively high metabolic half life and a relatively low clearance rate in the body.

50. (new) An agent as claimed in claim 45, wherein the proteasome inhibitor

a) is isolated in natural form from microorganisms or other natural sources; or

b) is formed from natural substances as a result of chemical modifications; or

c) is prepared completely synthetically; or

d) is synthesized *in vivo* using gene therapy methods.

51. (new) An agent as claimed in claim 50, wherein the proteasome inhibitor belongs to the following substance classes:

a) naturally occurring proteasome inhibitors:

- peptide derivatives which contain epoxyketone structures C-terminally,

- β-lactone derivatives,

- aclacinomycin A (also termed aclarubicin),

- lactacystin and its chemically modified variants, such as the cell membrane-penetrating variant "clastolactacystein β-lactone"

b) synthetically prepared proteasome inhibitors:

- modified peptide aldehydes, such as N-carbobenzoxy-L-leucinyl-L-leucinyl-L-leucinal (also designated MG132 or zLLL), its boric acid derivative MG232; N-carbobenzoxy-Leu-Leu-Nva-H (designated MG115; N-acetyl-L-leucinyl-L-leucinyl-L-norleucinal (designated

LLnL) and N-carbobenzoxy-Ile-Glu(OBut)-Ala-Leu-H (also designated PSI);

c) peptides which carry an α,β-epoxy ketone structure C-terminally, and also vinylsulfones, such as carbobenzoxy-L-leucinyl-L-leucinyl-L-leucine-vinylsulfone, or 4-hydroxy-5-iodo-3-nitrophenylacetyl-L-leucinyl-L-leucinyl-L-leucinevinylsulfone (NLVS)

d) glyoxylic acid or boric acid radicals, such as pyrazyl-CONH(CHPhe)CONH(CHisobutyl)$B(OH)_2$) and also dipeptidyl boric acid derivatives, or

e) pinacol esters, such as benzyloxycarbonyl(Cbz)-Leu-Leu-boroLeu pinacol ester.

52. (new) An agent as claimed in claim 50 wherein the particularly suitable proteasome inhibitor is the epoxyketone epoxomicin (epoxomycin, molecular formula: $C_{28}H_{86}N_4O_7$) and/or eponemicin (eponemycin, molecular formula: $C_{20}H_{36}N_2O_5$).

53. (new) An agent as claimed in claim 50 wherein the particularly suitable proteasome inhibitor is selected from the PS series

a) PS-519 as β-lactone, and also as lactacystin derivative the compound IR-[1S,4R,5S]]-1-(1-hydroxy-2-methylpropyl)-4-propyl-6-oxa-2-azabicyclo[3.2.0]heptane-3,7-dione - molecular formula $C_{12}H_{19}NO_4$ – and/or

b) PS-341 as peptidyl-boric acid derivative the compound N-pyrazinecarbonyl-L-phenylalanine-L-leucine-boric acid - molecular formula $C_{19}H_{25}BN_4O_4$ - and/or

c) PS-273 (morpholine-CONH-(CH-naphthyl)-CONH-(CH-isobutyl)-B(OH)2) and its enantiomer PS-293 and/or

d) compound PS-296 (8-quinolylsulfonyl-CONH-(CH-naphthyl)-CONH(-CH-isobutyl)-$B(OH)_2$) and/or

e) PS-303 (NH_2(CH-naphthyl)-CONH-(CH-isobutyl)-$B(OH)_2$) and/or

f) PS-321 as (morpholine-CONH-(CH-naphthyl)-CONH-(CH-phenylalanine)-$B(OH)_2$); - and/or

g) PS-334 (CH_3-NH-(CH-naphthyl-CONH-(CH-isobutyl)-$B(OH)_2$) and/or

h) the compound PS-325 (2-quinol-CONH-(CH-*homo*-phenylalanine)-CONH-(CH-isobutyl)-$B(OH)_2$) and/or

i) PS-352 (phenylalanine-CH_2-CH_2-CONH-(CH-phenylalanine)-CONH-(CH-isobutyl)1-$B(OH)_2$) and/or

j) PS-383 (pyridyl-CONH-(CH*p*F-phenylalanine)-CONH-(CH-isobutyl)-$B(OH)_2$).

54. (new) The use of proteasome inhibitors as claimed in claim 44 for inhibiting at least one of the entry/internalization process, the replication and the maturation and release of Flaviviridae.

55. (new) The use of proteasome inhibitors as claimed in claim 54 for inhibiting late processes in the Flaviviridae life cycle.

56. (new) The use as claimed in claim 54, wherein the proteasome inhibitor blocks to a large extent or completely prevent the production of infectious virions from Flaviviridae-infected cells.

57. (new) The use as claimed in claim 54, wherein the proteasome inhibitor causes inhibition of the release of virions and also reduces the infectivity of the virions which are released.

58. (new) The use as claimed in claim 54, wherein the proteasome inhibitor suppresses virus replication and thus the spread of an infection in vivo, i.e. in the liver tissue of an infected patient in the case of hepatitis C virus.

59. (new) The use of proteasome inhibitors as claimed in claim 54 for inhibiting the replication of Flaviviridae in accordance with the following mechanisms

a) blocking/reducing the release of new virions;

b) blocking/reducing the infectivity of released virions;

c) blocking/reducing the spread of infection in cultures of host cells;

d) blocking/reducing the spread of infection in infected organs *in vivo*.

60. (new) The use of proteasome inhibitors as claimed in claim 54 for suppressing flavivirus infections and pestivirus infections in humans and animals.

61. (new) The use of proteasome inhibitors as claimed in claim 54 for inducing the death of hepatocarcinoma cells.

62. (new) The use of proteasome inhibitors as claimed in claim 61 for suppressing and/or preventing the development of liver cell carcinomas.

63. (new) The use of proteasome inhibitors as claimed in claim 62 for treating patients who have established liver cell carcinomas.

64. (new) The use of proteasome inhibitors as claimed in claim 61 for treating/controlling/preventing HCV-induced liver cirrhosis and/or HCV-induced liver cell carcinomas, medicament-induced liver carcinomas, genetically determined liver carcinomas, environmentally determined liver carcinomas and/or liver carcinomas which are determined by a combination of viral and nonviral factors.

65. (new) The use of proteasome inhibitors as claimed in claim 61 for selectively eliminating liver carcinoma cells which develop as the result of a HCV infection, or a corresponding coinfection with HCV and hepatitis B virus (HBV), or a hepatitis delta virus (HDV)/HBV/HCV coinfection, human immunodeficiency virus (HIV)/HCV coinfections, or HCV and coinfections with other viruses, bacteria or parasites.

66. (new) The use of proteasome inhibitors as claimed in claim 61 for preventing the development, growth and metastasis of liver cell tumors and for preferentially destroying liver carcinoma cells in HCV-infected patients.

67. (new) The use of proteasome inhibitors as claimed in claim 54 for modulating the expression, modification and activity of the tumor suppressor protein p53 and other tumor suppressor proteins which are of importance in connection with hepatocellular carcinomas (HCCs).

68. (new) The use of proteasome inhibitors as claimed in claim 54 for liver cell regeneration in patients suffering from hepatitis.

69. (new) The use of proteasome inhibitors as claimed in claim 54 for regenerating patients following flavivirus infections.

70. (new) The use of proteasome inhibitors as claimed in claim 54 for regenerating stabled animals following flavivirus or pestivirus infections.

71. (new) The use of proteasome inhibitors as claimed in claim 54 for reducing the number of infected virus-producing cells in liver cell tissue.

72. (new) The use as claimed in claim 54 wherein the proteasome inhibitors alter the post-translational modification and proteolytic processing of Flaviviridae structural proteins and reduce the ability of the virus envelope proteins to dimerize and thereby reduce or block the release and infectivity of Flaviviridae.

73. (new) The use of proteasome inhibitors as claimed in claim 54 for inhibiting both the maintenance and persistence of a previously established infection and of a secondary infection including blocking the spread of a Flaviviridae infection in vivo.

74. (new) The use of more than one proteasome inhibitor as claimed in claim 50 in combination for the purpose of treating and controlling HCV-induced hepatitides, flavivirus-induced fever, hemorrhages and encephalitides and pestivirus-induced diseases.

75. (new) The use as claimed in claim 74 in combination with therapeutic agents which are already used in the antiviral therapy of Flaviviridae infections.

76. (new) The use as claimed in claim 74 for treating coinfections with different flaviviruses and pestiviruses.

77. (new) The use as claimed in claim 74 for treating coinfections of HCV and immunodeficiency viruses HIV-1 and HIV-2.

78. (new) The use as claimed in claim 77 for treating HCV/HIV coinfections in combination with HAART therapy.

79. (new) The use of proteasome inhibitors as claimed in claim 54 for preventing a reinfection with HCV in connection with liver transplantations and other organ transplantations.

80. (new) The use of proteasome inhibitors as claimed in claim 54 for preventing a reinfection with HCV in connection with cell therapies, by means of administering the agents before, during and after the transplantation.

81. (new) The use of proteasome inhibitors as claimed in claim 54 for preventing a reinfection with HCV in connection with the transplantation of virus-free organs to chronic virus carriers who still possess residual virus and can infect new organs and also in connection with the transfer of virus-containing organs from donors to virus-free patients.

82. (new) The use of proteasome inhibitors as claimed in claim 54 for preventing the establishment of a systemic Flaviviridae infection immediately following contact with infectious virus.

83. (new) The use of proteasome inhibitors as claimed in claim 54 for preventing a Flaviviridae infection in individuals who are at a high risk of fresh infection.

84. (new) The use of proteasome inhibitors as claimed in claim 54 for decreasing or eliminating a hepatitis by means of immune system-mediated mechanisms.

85. (new) The use of proteasome inhibitors as claimed in claim 50 for producing agents and/or pharmaceutical preparations for inhibiting the release, maturation and replication of Flaviviridae.

86. (new) The use of proteasome inhibitors as claimed in claim 85 for producing pharmaceuticals for the treatment and prophylaxis of HCV-induced hepatitides, flavivirus-induced fever, hemorrhages and encephalitides and pestivirus-induced diseases.